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An aqueous extract of *Platycodi radix* inhibits LPS-induced NF-κ B nuclear translocation in A549 cells

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We investigated the effects of aqueous extract from *Platycodi radix* (AEPR), a traditional drug used to treat acute lung inflammatory disease, on lipopolysaccharide (LPS)-induced inflammation in A549 human cultured airway epithelial cells. Nuclear factor-κB (NF-κB) and its inhibitory regulator, inhibitory-κB (I-κB), play crucial roles in LPS-induced inflammatory response. We show that LPS-induced nuclear translocation of NF-κBp65 is inhibited by AEPR. LPS-induced expression of I-κBα, which is expressed by LPS-induced activation of NF-κB, is inhibited by AEPR as well. Beside LPS-induced expression of a group of genes, such as tumor necrosis factor-α (TNF-α), inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2), are repressed by AEPR. We also found that expression of heat shock protein 70 (Hsp70), which has an anti-inflammatory activity, is increased by AEPR plus LPS. These results suggest that AEPR may act as a therapeutic agent for inflammatory disease through regulating the activity of NF-κB and expression of inflammatory genes.